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SECOND SERIES VOL. 10, No. 9

BULLETIN

OF

THE NEW YORK ACADEMY OF MEDICINE

INCORPORATED 1851

SEPTEMBER, 1934

PUBLISHED MONTHLY BY

THE NEW YORK ACADEMY OF MEDICINE

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New York, N. Y.

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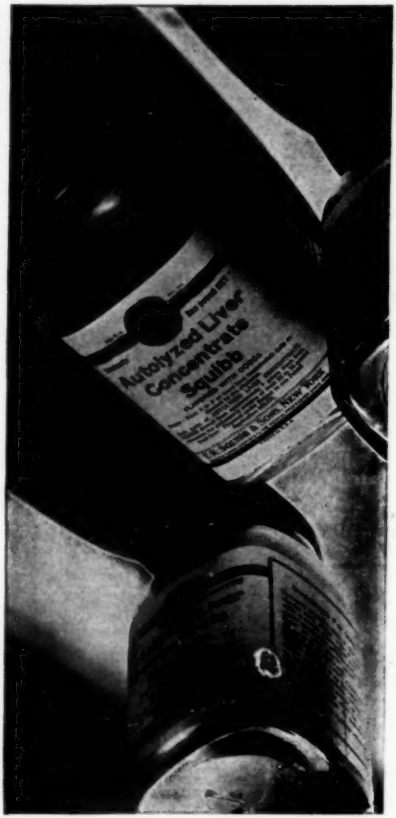

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EDITORIAL

THE MEDICAL HISTORY OF ROBERT SCHUMANN AND HIS FAMILY[1]

In the old Leipzig inn called The Coffee-Tree (Kaffeebaum), there sat of evenings, through the thirties of the 19th century, a pleasant mannered young fellow, of handsome appearance, attractive mien and enthusiastic nature, who reigned, in his chosen corner of this charming hostelry, as the leading spirit of a new departure in music. This was Robert Schumann, a man of unique genius, destined to become the prime-mover of music under the romantic tradition, the ablest critic of his art in his period, and one of the best beloved of all the great composers. Well-born, well-nurtured and well bred, he came of a family of Thuringian landowners, who settled eventually in Saxony. His paternal line numbered several clergymen, but in the Leipzig of Schumann's prime, which had supplanted Frankfurt as the center of the Continental book-trade, most of his people had gone over to the book business. His father was, in fact, a clergyman turned book-seller, a pursuit which had been followed by all his maternal uncles. His mother, the daughter of a Bohemian surgeon, had latent in her composition, no doubt, the Czech passion for music; for there is no evidence of any musical interest whatever in the Schumann strain. If there be anything in the Goethe-Scho-

[1]Read at meetings of the Johns Hopkins Medical History Club, February 15, 1932; the College of Physicians of Philadelphia, October 10, 1932; the Charaka Club (New York), November 15, 1933, and the Goethe Society, Baltimore, Md., January 20, 1934.

penhauer view of the inheritance of mental traits,[2] Schumann's musical genius came from the spindle side of his house. In one member of a collateral branch of the Schumann family, there was a solitary instance of suicide. The young Schumann himself was of jolly, loveable, easy-going nature, a natural gentleman of refined disposition and artistic tastes, bred to the law but inclined to neglect it, out of his unbounded passion for music, which had singled him out, as a Mendelian sport, among the solid, respectable, withal prosaic sibs of his paternal and maternal stock. A dispute with his widowed mother as to his fitness for the musician's calling was inevitable, and after some argument, he was permitted to take up advanced piano technique and composition under Friedrich Wieck, at that time one of the leading music teachers of Germany. Wieck, a man of bigoted, pedantic character, with an effective pedagogic method, proved an able instructor withal, whose Spartan rigors of discipline were visited mainly upon his young daughter Clara, with a single eye to making her a great pianist. As sometimes happens, in training musical talent of an unusual order, fortified by initial firmness of character, he succeeded, as did Mozart's father, beyond the dreams of avarice. At the age of nine, Clara was playing Mozart's E flat concerto to the delight of a Leipzig audience and, two years later, made her first public appearance in the exclusive concerts at the Gewandhaus. For this she received 30 thaler, of which 20 were turned over to her watchful parent. About the same time, there appears in her diary the first mention of a certain Herr Schumann, "who has been living with us as a pupil in music since Michaelmas." The two young people are soon rollicking playmates, as well as fellow students in counterpoint under Wagner's teacher, Theodor Weinlig, the old cantor of the Thomasschule. At thirteen, Clara is a sprightly, cheerful, dark-eyed, little elf, so ignorant of the life around her that

---

[2] *Vom Vater hab'ich die Statur,*
Des Lebens ernstes Führen,
Vom Mütterchen die Frohnatur
Und Lust zum Fabuliren.
Goethe.

she cannot tell ducks from geese, albeit her maiden effort in composition, a processional of swans (*Schwäne kommen gezogen*), was launched at the age of eleven. Even so, she has already known sorrow, through the separation of her parents, and is destined to further experience of it when she learns of her friend's engagement to the daughter of a Bohemian nobleman; for Schumann's mother, with an intelligence unusual in the maternal mind, had already drawn the child to her with the hint: "You must some day marry my Robert"; and was thus dubious about the ill-considered Bohemian engagement, soon destined to be broken off. At sixteen, Clara is an attractive, active-minded brunette of gentle features, strong compelling eyes, sensitive mouth, friendly, bitter-sweet smile, gracious mien and unstudied ease of manner and movement; of whose playing the great Goethe observes that "this girl has more strength than six boys"; and Mendelssohn that she rendered his B flat Capriccio "like a little devil." In brief, a very genuine, upstanding bit of piquant femininity, liked by everybody, and whose engagement to her old playmate is now a foregone conclusion. About this engagement to a man nine years her senior, Papa Wieck is nowise enthusiastic. He regards the making and breaking of Schumann's earlier engagement, humiliating to his daughter in itself, as an index of lack of character; questions Robert's ability to support a wife and grudgingly withholds consent until the fantastic youth can produce legal evidence to that effect. This leads to a protracted engagement, with many ups and downs, culminating in a lawsuit, which brings the sentimental relation of Robert and Clara into pitiless publicity all over Europe. At this time, Schumann had already made his mark as one of the most promising original and gifted composers of romantic Germany; but he had, at the same time, annihilated his future as a concert pianist by abuse of a mechanical contrivance designed to strengthen the weak fourth finger, but impairing its efficiency forever. The lawsuit is eventually won and the young couple are married in 1840; but Schumann's outlook on life is already clouded by morbid introspection, effect of worry over the publicity

attaching to his engagement and the permanent handicap of his finger dexterity, which gives to his piano music a certain strange physiognomy. That the impact of worry of this kind, upon a nature essentially noble-minded, may superinduce the autistic or shut-in life, is beyond question. Nevertheless, this turns out to be one of the happiest marriages ever made between people of artistic endowment, lasting without a break, up to the composer's untimely death, and the activator, in his case, of sixteen years of most astounding productivity. Masterpiece after masterpiece he pours out, with a facility rivalled only by the great of old; and the source of his inspiration is ever his wife, whom he surprises from day to day with some novel stroke of genius. The quality and originality of this music make it a thing apart. As with Rheingold or Tristan, it is like nothing ever heard before; and was destined to exert a lasting influence upon the later men, from Brahms and Richard Strauss to Jensen, Kirchner, Grieg, Dvorak or even Debussy. From such clever initial *pastiches* as the Papillons or the Carnevale, Schumann pyramids with ease up to such masterpieces as the C major Fantasie, the piano concerto, the piano quintet (a glorification of his artistic life), the songs, the Peri, the Manfred, the Spring and Rhineland symphonies; and these things, among the finest expressions existent of the inner life of humanity, have enshrined the memory of the composer with an aureole of affection, comparable with that accorded to Oliver Goldsmith or Charles Lamb in English literature. To know the music is to love the man. The gentle prime-mover of all this enchantment is Clara, who, like Minna von Barnhelm, prides herself upon her Saxon strength and fidelity (*ein starkes Mädchen*), becomes, in fact, the business manager of this household, which she finances by teaching and giving concerts, while Robert dreams away his days at the piano or in his Kaffeebaum corner, reluctant even to make himself known to the extent of accompanying his wife on concert tours into Russia or the Netherlands. An inspiring writer, an influential journalist, a discerning critic, he is but an indifferent orchestral director, prone to lose the

count and dislocate rhythm by lapsing into reverie; and so loses his position in Düsseldorf. This and other setbacks begin to prey upon his mind. For the first time in his married period, this gentle housefather scolds his devoted wife, an abrupt change, noted even in the Hippocratic Canon as ominous. As time wears on, Schumann becomes more and more sensitive and autistic, inclined to live within himself and sit mumchance, without uttering a syllable, in social gatherings. Presently there develops a strange humming in the ears, upon a single note, intensified and prolonged up to excruciating agony, with insomnia, set off by audition of music more heavenly than he had ever dreamed. On a fateful morning (February 27, 1854), he rushes from the house in scanty attire and throws himself into the Rhine. His life is saved by the captain of a nearby vessel and he is brought back home by masqueraders, returning from nocturnal festivities. He is pronounced insane, and is committed to asylum, living on for two years, with rare lucid intervals, to die on July 29, 1856 at the age of forty six. The autopsy shows, among other things, certain spicules of cranial bone, impinging upon the brain and lacerating the cerebral membranes. The brain is one of the smallest and lightest on record in the medical histories of men of genius.

Fortunately for Clara, there had appeared, just upon this grim break-up in her life, a young spirit, stronger even than herself, who was providentially fated to be the friend in need and her bulwark against despair. Brave with the courage of the gentle (the faithful Mendelssohn long dead), the unhappy woman now faces life alone, with the additional responsibility of supporting a large family, but not entirely unfriended. Johannes Brahms, whom Schumann, with the insight of true genius had hailed as the coming composer, the continuator of the great Bach-Beethoven tradition, is at this time a light-hearted, blond, blue-eyed boy, long haired, like *crinitus Iopas* in Virgil, touring the Rhineland in search of opportunities, with hardly a penny in his pocket. He happens in upon the Schumanns unannounced, if not entirely unbidden. At the piano he astounds

them with the virile fire of his execution and the originality of his conceptions. As he plays, his young Raphael face is transfigured with the light that never was on land or sea. In the dire poverty and hardship of his childhood, he is like unto Schiller.[3] He has even eked out a living on occasion by playing in the lowest sailor's dives; but the sea-air of Hamburg is in his veins and he is already winning up to manhood unscathed. His gratitude to his new friends is unbounded and through the long tragedy of Schumann's illness and beyond it, he stands by the stricken family, *"als kleiner Mann,"* collaborating with Joachim in getting up concert tours for Clara, albeit penniless himself. Naturally the boy falls in love with her, now a handsome woman in her prime; but here, his mordant sense of humor collides with his feeling of utter helplessness and sorrow over the tragedy of Schumann's fate, to produce the spiritual conflict which he welds into his great D minor concerto. By this token, Brahms attains to the sterner stuff of which manhood is made, surely one of the best men who ever lived upon earth. Clara takes up concert touring in England and becomes at once a perennial favorite with the English people, to whom her sadness seems "so pitiful." Like the devoted mother in the Spoon River Anthology, she raises her little brood up to the self-supporting status, ranking with Essipoff and Sophie Menter as a concert performer, up to her retirement (aet. 59), as teacher in a conservatory at Frankfort on the Main. Her essential service is that along with Brahms and Joachim, she has made the better sort of music a going concern on the concert stage, supplanting the vapid Liszt-Thalberg tradition of meretricious variations, operatic fantasias and salon music of affected mien. The native fire, which had rivetted Goethe and Mendelssohn, remains the essential trait of her being to the last. Even to a late American observer, she seems, in playing Bach, as if "crowned with diamonds." Brahms remains

[3]As commemorated in the verse of Emanuel Geibel:

"Ein armes Dach nur war's im Gau der Schwaben,
Dort wo der Geniusengel segnend eingekehrt,
Der Sorge Wohnsitz die den blonden Knaben
Früh lehrte wie man duldet, kämpft, entbehrt."

the faithful friend and advisor of the family to the end, surviving her death by less than a year.

Eight children were born to the Schumanns, of whom one died in infancy. The fate of the remaining seven is of considerable interest, as a phase of the biological investigation of family histories (*Familienforschung*), which is now a going concern in Germany.[4] Genealogy and family-trees, from the tables of Egyptian and Sumerian Kings to Burke's Peerage or the Almanach de Gotha, are as old as the hills; but the pace in the utilization of family records was set by the novelists. A favorite theme of the English masters of fiction, from Richardson, Fielding and Smollett to John Galt, Thackeray (The Newcomes) or Galsworthy (Forsyte Saga), has been the life-history of one or more familial groups, centering around the fortunes or adventures of some particular member. This plan was carried out on a grand scale by Balzac, whose *Comédie humaine* covers a whole cross-section of French society in his period; and latterly by Zola (Rougon-Macquart series) and Marcel Proust (*À la recherche du temps perdu*). Familiar examples of more recent vintage are Thomas Mann's Buddenbrooks, Hergesheimer's Black Pennies and The Orissers by L. H. Myers.

Assuming that Schumann's genius was the resultant of a favorable constellation of Mendelian factors and that Clara came by her pianistic talent through her father, one would expect a natural outcropping of musical ability in the children. As a matter of fact, none of the Schumann children had any particular musical gift. Such ability in piano playing as the daughters possessed was largely environmental, the result of careful training given them by the mother and by Brahms. The working out of Mendelian principles in the little family is exhibited along quite different lines and in a significant and striking way. Of the Schumann children:

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[4]For the genealogy of the Schumann family, see: Stammbaum Robert Schumanns. *Veröffentl. d. Schumanngesellsch.*, Zwickau, No. 3. For the life-histories of the eight children, *see*, Memoirs of Eugenie Schumann, London, W. Heinemann, 1927.

1. Marie, the eldest, born on September 1, 1841, contravenes the theories of Karl Pearson and Still about the handicap of the first-born. Far from being a weakling, she was the strongest and most enduring of the entire family, in every respect the flower of the flock. Tall, upstanding, thoughtful, serious, dignified, she inherited the mother's fine dark eyes, an index of her firm, capable character. Endowed with no outstanding musical talent, she became by dint of application, a very capable pianist, often assisting her mother in concert performances for four hands or two pianos. As being the eldest child, and her father's favorite, she was destined to be her mother's regimental adjutant and handy man in running the family after the great disaster. "In spite of your equanimity," said her sister, "you had a distinct tendency to *furor teutonicus,* which would break like a thunderstorm and as suddenly pass. You were also the thoughtful one of the family . . . You were never really happy unless you took care of our mother and us from morning till night, and of many others as well." Strong and sturdy, Marie surpassed her mother's longevity of 76 by 14 years, dying at the age of 90.

2. Else, the second child, born on April 25, 1843, was tall, slender, strongly built, blue-eyed, fair-haired and strong featured. She inherited her mother's sturdy independence, self-confidence, self-possession and tendency to strike out for herself. She left the family early to teach music upon her own, and while possessing no more native talent than thousands of cultivated amateurs in Germany, she became a very competent pianist, capable of replacing her mother in chamber music on the slightest notice. In 1877, she married Louis Sommerhoff, a prosperous American merchant, had four sons and a daughter, and in 1927, was still living at the age of 85. The names of her descendants may be found in the New York City Directory.

3. Julie, born March 11, 1845, was blonde and perfectly normal, like all the other daughters. In 1868, she married an Italian nobleman, Count Marmorito, had two children and died in childbirth in 1872 (aet 27).

4. Emil, born February 8, 1846, died in 1847 of glandular fever.

5. Ludwig, born, January 20, 1848, was dark and strongly built, like his mother, but gloomy, sensitive, introspective and clumsy in movement. He became insane at the age of 22, was confined to asylum for 31 years and died in 1900 (aet. 52). Here the Mendelian recessive in the Schumann strain became dominant, duplicating the father's fate.

6. Ferdinand, born July 16, 1849, was dark-haired and blue-eyed and inherited his mother's practical business ability. Educated at Berlin, he went into business early, became a bank official and married the daughter of a landed proprietor. Military service in the winter campaign of the Franco-Prussian war brought on a rheumatic seizure, intensified during militia service ten years later. Committed to hospital in Berlin, he acquired the morphine habit through the carelessness of an unscrupulous doctor, lost his health and his competence, and died in 1891 (aet. 49), leaving six children on Clara's hands.

7. Eugenie, born December 1, 1851, resembled the mother in sturdiness and competence. She became a music teacher, wrote the memoirs of her family, and is still living at the age of 83.

8. Felix, born June 11, 1854, shortly after his father's break-down, was good-looking, attractive, of artistic bent, author of poems which Brahms set to music, and himself determined to be a musician. The development of lung trouble precluded this possibility and he took up law. Passing his examinations brilliantly, he became a dashing Heidelberg student in cerevis, riding habit and boots, but died prematurely of tuberculosis at 25.

Of the four girls of the Schumann family, three (Marie, Else, Eugenie) lived longer than their mother and it is possible that Julie, who died in childbirth, had the same superior longevity *in posse*. Of the four boys, three (Emil, Ferdinand, Felix) died early of those intercurrent dis-

eases which, in the phrase of Chalmers of Glasgow, "strike laterally," while Ludwig was singled out as the victim of vertical incidence of a Mendelian recessive (insanity). In respect of longevity, the girls were in the trend of the mother's biological fate and even outpaced it; the boys were, all of them, shorter lived than the father.

It remains to say something about the declining years of the two people who made the raising of these seven children possible. The mother, a good disciplinarian, lived among her children as a kind of big sister, signing her letters to them *Mutter Clara*: in other words, she had grasped the true military significance of leadership and "discipline," not to bully but to make them disciples of Clara. In piano training, she followed her father's device, to get a beautiful intonation from the keyboard by pressure rather than by percussion. Brahms was a pioneer in loosening the stiff wrist, with a single eye to autonomy of the ten fingers as individual voices, say in an intricate Bach fugue. Some of the puzzle-headed exercises which he wrote out for the Schumann children were later incorporated in his *51 Uebungen*. The hardships of his childhood and youth, the icy coldness with which music so original as his own was at first received, superinduced in Brahms the usual defensive reaction of a sensitive nature:[5] he was occasionally brusque under gratuitous criticism, even with Madame Schumann; for which he was sometimes vigorously rated by her young daughters. But for Brahms, life veritably began at forty, when his experimental period of constructive composition was by and he was already branching out into genuine creative effort. By this time, Clara's financial status, as a self-supporting bread winner of her household, was assured and Brahms himself was beginning to bristle with the touchy independence of the confirmed bachelor, very much inclined to have (and go) his own way. The masculine-minded youth of gentle face had merged into a sturdy, thickset being of Jove-like head,

[5]In his Viennese period, when he was just the least little bit spoiled or surfeited by adulation, Brahms sometimes regretted that audiences "no longer hissed his compositions."

expansive beard and intensely masculine outside, with unsuspected sources of tenderness and gracious compassion within. Of an early picture of himself, Brahms observed: "At that time, I looked like a dubious candidate in theology." As a boy, he had virtually adopted the Schumann family and made himself a member of it. As the years go by, his manner toward Clara becomes that of a devoted son. The only differences between the two friends, as a rule, turn upon her criticisms of certain novel effects of his music, which do not square with her old fashioned sense of the fitness of things. In old age, the atmosphere about either is sometimes thunderous; effect of the ponderous pragmatism of the Nordic approach, which somehow visualizes Henry James' Italian marquis in London, waving his cane in the air and hissing: "They have *heavy* minds."[6] But it is through this unfailing high seriousness that the music of Brahms attains the heights of "splendor and magnanimity" noted by an English writer. Of impeccable structure, it has extraordinary power, not only to elevate and sustain us over the dead spaces of life, but even to exalt and ennoble. In ordinary human relations, often with Americans, Brahms was himself one of the most charming of men, and this, with all due allowance for the many anecdotes concerning his offhand bouts of old bachelor rudeness. He left one social gathering with the mocking query: "Is there anyone in this room whom I have *not* insulted?"; and a well-known silhouette represents him trudging through the streets of Vienna with an attendant hedgehog. As "there is no animal in nature so patient as the fretful porcupine," it is possible that Brahms was sometimes teased to get a "rise" out of him. Some jolly Viennese girls, who were trundling him over the ice in a little sleigh, once tipped him into a snow-bank, no doubt to hear the old bachelor curse and swear. In making a generous subscription for a prospective monument to the Swiss composer Raff, Brahms could not resist the sally: "Get that statue up quickly before the composer himself

[6] In the Princess Casamassima, à propos of the Anglo Saxon, and general Nordic talent for fumbling.

is forgotten"; and it delighted him to silence cheap flattery in the open by asserting (with uplifted forefinger) that some blatant bit of band music, then playing, was one of his own compositions. On the other hand, even his dressing table was usually cluttered up with toys to present to children on his walks; he gave his poverty-stricken father, for reference in distress, a book of religious character, which was later found to be filled with bank-notes; and to a poor waiter, bullied by a bounder, he handed a double *douceur,* whispering: "That is for the other gentleman also." In brief, a man of essentially noble nature, with occasional lapses into the sly malice of the physical dwarf (*Kleine Leute haben grosse Herzen*). Loathing adulation and all manner of lionizing, Brahms side-stepped invitations to visit England, "to make his works better known," with the dry reminder: "They are already printed for that purpose." An offer of a liberal honorarium for a new composition, "to order," was rejected with the hint that some slight attention be paid his already published works. His jeers at the trite quality of Highland reels were set off, perhaps ironically, by such "hauntings of Celtism" as his three versions of "Edward, Edward," Murray's *Ermordung,* the beautiful intermezzo on Lady Ann Bothwell's Lament (opus 117, No. 1) or his effective use of the primitive pentatonic scale of Scotland (*c, d, e, g, a*) in the andantino of the Clarinet Quintet—

"He's pu'd the rose o' English loons,
And brok'n the harp o' Irish clowns,
But our Scotch thistle will jag his thumbs,
This wee, wee German Lairdie."

Brahms' pawky humors, applied as freely to himself as to others, had, in fact, a kind of "Scotch snap" to them.

Like most men of marked originality and strong intellectual bias, Brahms was, in brief, a bundle of contrary and contradictory traits, spontaneous, whimsical and therefore unpredictable, perhaps undecipherable, even to himself. A child of the people, a patron of the folk-song, he could compose music of essentially aristocratic type, such as the violin sonatas, the intermezzo for piano or the

clarinet quintet. When the University of Breslau gave him a doctor's degree as "the master of austere music," he acknowledged the diploma on a postal card and sent in, as his inaugural dissertation, a rollicking Academic Overture on German student songs. He stood up for Beethoven's imitation of a music box in the Emperor Concerto, admired the banjo and, as the great master of polymorphic rhythms, thought even of utilizing "not the stupid tunes, but the interesting rhythmus of ragtime" (Schauffler). Did he beam with satisfaction over a new performer or a new composition, the goose of either was cooked in advance; if he looked sad and serious and sometimes left the room, he would go any length to help performer or composer. Ordinarily modest and reticent as a mouse in a corner about his own work, woe to the luckless pretender who came within range of his biting tongue! Abstemious as a miser with regard to his own creature comforts, he squandered to a fault in helping the poor and needy, supported several indigent composers or their relatives, lifted Dvorak out of obscure poverty into success, corrected his proofs and twice placed his entire fortune at his disposal. If the same whimsicality made him sometimes churlish in genteel interiors, Brahms would put himself to endless inconvenience to spare or help the forlorn and down-trodden, and actually pretended to be a Roman Catholic in Italy, out of a Quixotic consideration for a people he liked. Certain negligible eccentricities in dress and behavior he attributed to the chill penury of his boyhood, the cruel necessity which compelled a virtual child to gain his living in the haunts of vice.[7]

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[7]It is well to bear in mind that the lot of great composers in the past was not even as secure and respectable as that of hired servants today. Sebastian Bach was of less social consequence in his community than the court functionary who trimmed the toe-nails of His Serene Highness. Mozart and Haydn were virtual chattels of great personages, uniformed, like organ-grinders' monkeys, sometimes even composing music to order. Mozart, divine child of genius, fed at the servant's table, was starved by patrons, cheated by publishers, and once even kicked downstairs by an archbishop's lackey. Some of the most beautiful songs ever conceived by mortal man were composed by Schubert out of hand to pay off his beer

Up to the age of 24, Brahms had remained a beardless boy, *presqu' un éphèbe,* of seraph face and piping, high treble voice. The change to the burly, thickset, vigorous, heavily bearded man of his prime was accomplished by a corresponding change in his physiological life, which any doctor would understand, about which lengthy chapters have been published and over which any man of the world would draw a charitable veil. He pursued no neighbors' wives, daughters, sisters, cousins or aunts; indeed so unquestioned was his veneration for the married state that he terminated his lifelong friendship with Joachim, when the latter came to divorce his wife. Of a prospective *visite de noces* with a much married pianist, Brahms observed: "He is sure to marry several times again and [with kind permission] I'll just skip meeting wife No. 3." It is doubtful if Brahms' persistent backing away from the married state was anything more than the usual fugitive slave reaction of the independent artist, as in the case of Artemus Ward and the Mormon widows or of Liszt, when pursued to the very portals of the Vatican by *Fürstin Hinterlist*: "Gregory VII was a great philanthropist." In 1891, Brahms, the self-willed old bachelor, published the original version of Schumann's D minor symphony without the consent of his widow, who let loose upon him the vials of her wrath, even to a break in their oldtime

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scores in the little inn at Grinzing. Until late in the 19th Century, composer and virtuoso were regarded as performing dogs on show, who entered the house by the servant's staircase and were separated by a crosswise rope from the aristocracy. To the noble patron in an English novel, the term "musician" connotes long hair and dirty hands. Humiliations of this kind were weathered by the great composers of the past with a dignity and fortitude which are beyond all human praise. Rough handling made them rough customers, in accordance with Newton's Third Law of Motion. If today, the composer, the professional musician, have become respected and respectable, it is due to the superior social gifts of such modern men as Liszt, Chopin, Tchaikovski or St. Saens and to the upstanding independence of such dynamic personalities as Händel, Beethoven, Wagner and Brahms. For admirable research-work on the degraded status of music and musicians before the time of Wagner and of Wagner's enormous accomplishment in improving the situation, see Ernest Newman's recent life of Wagner, vol. I (New York, Knopf, 1933).

friendship. Brahms effected reconciliation by stressing his forty years of devoted service, his loneliness and the real point at issue: "Allow me to repeat that you and your husband represent the most beautiful experience in my life." Upon the strength of this simple, honest statement, we may dismiss, as idle, impertinent gossip, certain suppositions of recent biographers touching the fair fame of these two benefactors of the human race and doctored up, as usual, *ex post facto,* after the victims are dead and gone and no longer here to defend themselves. Were Brahms on hand just now, to encounter some of these prurient pickthanks, one can fancy him pouncing upon them with the agility, ability, ferocity and velocity of some of the big cats out of the tropical jungles, albeit not primarily a member of the cat family himself.[8]

The definite break in Brahms' otherwise robust health came at Clara's funeral, which he all but missed by taking the wrong train, and to which he hastened on foot in a driving rain, lingering by the grave in an extra bath of perspiration. A latent cancer of the liver was aggravated by the chill, and presently Brahms stands at the gates of death. In the virile *Totentanz* of his E minor symphony, he had greeted death in a jocund, exultant spirit. But now, his mood, as conveyed in one of his Serious Songs, is that of Odysseus in Hades: *O Tod! wie bitter bist du!.* Not only had he attained to serene satisfaction through his acknowledged position as the strongest and most dignified figure in modern German art, the last of the great line of classical composers; but latent within himself were untapped wellsprings of creative power, productive of such things as his terminal double viola quintet (opus 111), which is incandescent with genius of the highest order, incidentally a charming pendant to that radiant apotheosis of Viennese *Gemütlichkeit,* the Schubert Octet. To Brahms, in his au-

[8]Many of the above anecdotes and data are to be found in the 8 volumes of the Life of Brahms by Max Kalbeck and "The Unknown Brahms" by Robert Schauffler. There is no well-ascertained fact about Brahms which does not go to show, what his music and the extant portraits plainly tell us, that he was an inflexibly honest and absolutely reliable man.

tumnal period, came easy mastery of the most difficult material and a magical rejuvenation, giving to his F Major Symphony or his later songs and chamber music a life-enhancing freshness and fragrance—

"Over the winter glaciers
We see the summer glow,
And through the wild-piled snow-drift,
The warm rosebuds below."

To a man so inspired and endowed, death came as a tedious, troublesome, unwelcome visitor. Toward the end, Brahms resumes the past with Georg Henschel, and what he says will convey some notion of his ideals and of what he sought to express in music:

"How few true men there are in the world. The two Schumanns—Robert and Clara—there you have two beautiful *Menschenbilder.* Knowledge, achievement, power, worldly place—nothing can outweigh this, to be a beautiful *Menschenbild.*"

No slur which may be cast upon the memory of Johannes Brahms, by microscopic or myxoscopic minds, can outweigh the splendor and magnanimity of this parting tribute to the two human beings whose friendship had meant more to him than anything else in life.

F. H. GARRISON.

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HYPERPARATHYROIDISM AND ITS RELATIONSHIP TO DISEASES OF BONE*

HENRY L. JAFFE

A brief preliminary explanation of normal parathyroid function seems necessary for the subject that follows. The parathyroids when functioning physiologically, elaborate a hormone which is secreted in such quantities as to regulate in amount and maintain within certain levels the blood calcium and possibly phosphorus. Intimately allied with the parathyroids in their process of regulation through hormone production, are such other factors as the calcium and phosphorus available in the diet, the ratio of these substances to each other, and the vitamin-D availability. These seem to be important in determining the level of physiologic activity of the parathyroid glands. Considerable evidence is accumulating to the effect that the absence of parathyroid hormone can be satisfactorily compensated for by regulation of the calcium, phosphorus, and vitamin-D intake. Tetany, following ordinary parathyroidectomy in both man and animals, can be greatly ameliorated in severity or possibly even prevented by both high calcium intakes and sufficient vitamin-D therapy. Normally, then, the chief activity of the parathyroid gland is the production of a hormone that helps to equilibrate the calcium and phosphorus between the tissues and the blood.

Hyperfunction of the parathyroids, or hyperparathyroidism, but recently conceived and established, has come within the realm of actual knowledge only during the last eight years. While considerable progress has been made in the field of parathyroid hyperfunction, much still re-

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From the Laboratory Division, Hospital for Joint Diseases, New York.

mains to be elucidated. Hyperparathyroidism is now, on the one hand, synonymous with Recklinghausen's disease of bone. In this condition, a pathologic degree of hypersecretion has been proved to exist as the primary cause of the disease. Another question promptly arises here: Does hyperparathyroidism exist in other bone conditions than Recklinghausen's disease? In such diseases as rickets, osteomalacia, calcium deficiency osteoporosis, etc., where the parathyroid glands are sometimes found enlarged—is the enlargement an indication of a compensatory hyperactivity, a physiologic or secondary hyperparathyroidism? As yet, there is no definite means of proof to decide this problem.

Reverting once more to the hyperparathyroidism of pathologic degree, certain definite clinical and chemical manifestations signal the presence of parathyroid hormone secretion in abnormally great quantities. In the absence of serious complications derived from its occurrence, some of the more exact indications are: an increased serum calcium or hypercalcemia, a depressed serum phosphorus or hypophosphatemia, an elevated serum phosphatase, an increased excretion of calcium and phosphorus particularly through the urine, and—progressively, constantly—a decalcification of the bones.

Before proceeding, let us consider the general substance of this discussion. I propose to give: (1), a very brief survey of primary hyperparathyroidism or Recklinghausen's disease; (2), a review of experimental hyperparathyroidism, particularly stressing its connotations in relation to Recklinghausen's disease; (3), an examination of some other metabolic diseases of bone in their relation to Recklinghausen's disease and the possible role of hyperparathyroidism in their evolution; with, finally, (4), a discussion of the rationale and basis of the recent surgical popularity of parathyroidectomy in other bone diseases than Recklinghausen's.

First under consideration, we have Recklinghausen's disease, which has been previously indicated as the effect

of pathologic or primary hyperparathyroidism. As clinically encountered, it is usually the product of many years' more or less progressive evolution. Dependant upon the stage of its development when brought under observation, the pathologic changes in the osseous system may cover a wide range. The lesions may be so advanced as obviously to suggest the designation of generalized ostitis fibrosa cystica, or so slight as to be barely perceptible grossly. Between these two extremes are cases that evidence varying degrees and various stages of involvement. Thus, in a specific case, a long tubular bone may grossly appear entirely free of any involvement, while other long tubular bones present the more classic evidences of the disease:—marked porosity, cysts, giant-cell tumors, and an abundance of connective tissue, especially in the metaphyseal marrow cavities. Since, however, Recklinghausen's disease is a generalized one—which implies the subjection of the entire osseous system to its influence—histologic examination of the apparently uninvolved bone will, nevertheless, demonstrate microscopic indications of its presence.

Recklinghausen's disease generally evokes the image of cysts and giant-cell tumors as essential accompaniments of its pathologic features, whereas that is not necessarily so. Bones may undergo extensive resorption and transformation in the course of the malady, yet careful search will fail to disclose either gross or microscopic cysts or giant-cell tumors. Rather, the essential process due to pathologic chronic hyperparathyroidism in the evolution of the final pathologic picture is progressive demineralization; upon it, all other changes are consequent. The decalcification is the stimulus for the appearance of numerous osteoclasts with their Howship's lacunae and the proliferation of the marrow connective tissue. Finally, it is conceivable that the chronic hyperparathyroidism may incidentally, in the course of the development of bone changes, favor extensive hemorrhage into the marrow, resulting in brown blood cysts and the formation of giant-cell tumors. The pathologic picture may be modified by, and vary with,

the age of the patient, the duration of the disease, the intake and availability of mineral salts, the celerity and degree of decalcification, and stresses, strains and possible injuries to which the decalcified skeleton is subjected. As investigators are becoming more aware of the wide range of pathologic changes in the bones, produced by hyperparathyroidism, the hope draws closer to realization that the severe deformities, such as Recklinghausen originally described, by virtue of early recognition will be wholly eliminated.

The association of tumor-like, parathyroid enlargement with the bone changes of Recklinghausen's disease has become increasingly evident, especially since 1925, when Mandl made his important discovery. He was the first to recognize that immediate clinical improvement and regression of such bone changes occurs after the removal of an adenomatous, parathyroid tumor. This observation decided the question whether in Recklinghausen's disease the parathyroid abnormality was the result of the bone disease or its cause. A rapidly increasing series of cases, of which about 60 cases have since been reported, support the hypothesis that parathyroid hypersecretion is the basis for the inception of the bone lesions of generalized ostitis fibrosa cystica. In this country, further clarification of the clinical aspects of the subject was achieved principally through the work of DuBois, Barr, Boyd, Aub and their associates. In no other disease in which parathyroid enlargement has been encountered has there been like proof —as here—that there exists an actual hyperfunctioning of the parathyroid glands instigating bone changes.

The accumulated postmortem and clinical experience with Recklinghausen's disease has demonstrated variability in the number, size, weight, position, gross and histologic appearance of the offending parathyroid tissue. The examinations have shown that one or more benign, parathyroid adenomas, without any other parathyroid tissue, may be present, or an enlarged parathyroid with one or more (in some instances, four) normal-sized para-

thyroids, may be found. Thus, the likelihood of the absence of other parathyroid tissue indicates the danger of parathyroid tetany following operative removal of a benign parathyroid adenoma. As to size, the tumors have measured from 1 to 7 cm.; they have weighed as much as 35 gm. No correlation has been established between the size of the parathyroid tumor and the severity of the disease. In spite of their large size, it is amazing that so few of them have been palpated before operative removal; some of the largest glands were not palpable because they were tucked away between the esophagus and the trachea. Hypersecreting parathyroid adenomas have even been found completely within the substance of the thyroid gland, as well as in the mediastinum.

The parathyroid tumor is usually nodular and cystic; the cut surface appears yellowish; generally, where histologic semblance of normal parathyroid tissue remains, the tumor is found composed of principal cells. There have been described a few instances of fairly large parathyroid-like tumors without bone changes. It is very difficult to give any interpretation to these reports. The danger of confusing parastrumas of the thyroid with parathyroid adenomas, is only too obvious. Hereafter, in attempting to establish the parathyroid nature of such tumors, it will be necessary to prove the existence in them of the parathyroid hormone. Among the other pathologic manifestations are—metastatic calcification, particularly in the mucosa of the stomach, the lung and the kidneys; renal calculi are also commonly observed.

From the clinical standpoint, pathologic primary hyperparathyroidism is a disease but infrequently encountered in comparison with the other conditions causing extensive osseous decalcification. When such a patient does come for diagnosis, the indications and symptoms are found variable with each case. Diagnosis is often difficult to make; the clinician should consider and interpret: (a) the history and physical examination, (b) the chemical analyses and radiographs—always relating them to the

history and physical examination; finally, (c) he must consider differentially the other conditions that lead to decalcification and porosity of the bones and which may simulate Recklinghausen's disease. It is not my purpose here to discuss in detail the means of arriving at a diagnosis. This has been done in the many excellent clinical articles published. Suffice it for the purpose of this presentation to differentiate Recklinghausen's disease from certain others by reference to its more precisely individual characteristics.

We come now to the second consideration of this discourse:—a review of experimental hyperparathyroidism, with especial emphasis on its relation to clinical Recklinghausen's disease. With Collip's preparation of the active parathyroid extract, it has been possible to study hyperparathyroidism in the experimental state. In association with my colleagues, Doctors Bodansky and Blair, certain of its effects on animals have been demonstrated. Repeated, small, nontoxic doses of this extract, injected into dogs, soon raises the serum calcium and causes an increased excretion of calcium and phosphorus through the urine. The dog is so particularly sensitive to moderate dosage, as well as to overdosage, that unless the calcium intake of the animal is restricted or the dosage of parathyroid very limited, fatal toxic effects with marked hypercalcemia and hyperphosphatemia are likely to ensue. Dogs on either a liberal or low calcium intake, when treated for some time with parathyroid extract, show a regression of the initial tendency to hypercalcemia. On a low calcium intake, the regressing hypercalcemia may eventually be replaced by a hypocalcemia. Sometimes, such dogs die from the toxic effects of the parathyroid extract, without even evincing a terminal hypercalcemia. In dogs, with chronic experimental hyperparathyroidism, the serum phosphorus tends to remain at or to exceed the normal level, regardless of whether the calcium in the diet has been low or liberal. This hyperphosphatemia contrasts with the hypophosphatemia usually observed in clinical cases of

Recklinghausen's disease without nephritic complication. This, of itself, indicates the great sensitivity of the dog to injections of parathyroid extract.

On either the adequate or low calcium diet, the dogs with experimental hyperparathyroidism demonstrate bone decalcification and resorption, and reactive marrow fibrosis. These are also the basic histologic changes observed in the bones of clinical cases of Recklinghausen's disease. We have been led to conclude from our experiments that the dog has a much greater sensitivity to hyperparathyroidism than man. Whether the difference in their reactions is more apparent than actual, one cannot say. In the course of Recklinghausen's disease in man, much more delicate adjustments and compensations are probably made than in the dog.

However, other animals, by reason of their greater resistance to parathyroid extract, open other aspects of the problem. In the rabbit, the effects of parathormone, if elicited at all, are very slight. Hypercalcemia can be evoked by a single dose only when it is extremely large; toxicity, or overdosage effects as seen in dogs, is absent in rabbits given repeated, large, daily doses, when preliminary treatment with smaller doses has been administered. Furthermore, the injection of as much as 4,200 units, into a young adult rabbit, during 90 days of treatment, led to practically no bone changes.

The guinea pig, although relatively resistant, is much less so than the rabbit. It is possible to obtain in young guinea pigs, early in the course of repeated daily treatment with parathormone, hypercalcemia and hyperphosphatemia, decalcification and fibrous transformation of rapidly growing bones or portions of such bones. In young and adult guinea pigs, a compensation is established during prolonged treatment, which enables them to tolerate repeated, large doses, without hypercalcemia, and which permits considerable repair of bone lesions produced earlier in the treatment. Healing of the bone lesions in the guinea pig brings about a definite progression towards normal bone.

On the other hand, the rat, as we will presently see, has an altogether different healing response. The rat, whose bones are fairly reactive to parathormone, unlike the dog, tolerates relatively large doses without too great a risk of fatal hypercalcemia and hyperphosphatemia. Therefore, the rat can be given a high or adequate calcium diet, when injected with parathyroid extract. After short periods of treatment with relatively large doses, the bones of rats manifest all the usual changes of hyperparathyroidism:—pronounced decalcification and resorption, and the appearance of fiber marrow. However, it is notable that rats fed an adequate calcium diet and kept on parathyroid extract for long periods of time finally evidence the development of marked cortical thickening, and metaphyseal osteosclerosis. This osteosclerosis is to be interpreted as a superabundant healing phenomenon. The bone thickens because new-bone formation is excessive, but it occurs in the presence of continued bone resorption. Such rats are apparently in calcium equilibrium in spite of long treatment with parathyroid extract; this is one of the reasons for the peculiar healing response. The microscopic appearance of such sclerotic bone simulates, but does not exactly duplicate Paget's bone. The suggestion in that direction springs from the numerous cement lines somewhat chaotically disposed. This osteosclerosis reflects the mechanism of construction of sclerotic bone as seen in Paget's disease, or possibly Albers-Schoenberg's disease, although the etiologic circumstances are seemingly entirely different.

In our experimental studies of hyperparathyroidism, the mechanism by which bone decalcification and resorption are brought about, has always been of particular interest to us. The histologic study of a great number of decalcified and undecalcified bones, from animals suffering from experimental hyperparathyroidism, has led us to the conclusion that hyperparathyroidism (as a result of the changes that it induces in the tissue fluids circulating about the bone) has but one primary effect: That is, the solution of mineral salts out of the bone. This halisteresis

occurs at the surfaces of contact between the tissue fluid and the bone. All other appearances and effects are secondary to the production of minute and narrow zones of decalcification on the subperiosteal, subendosteal, trabecular and Haversian canal surfaces.

We thus come to the logical question: what has the animal experimentation taught us in relation to clinical Recklinghausen's disease? We should first recognize that complete duplication of the advanced form of this disease as seen in man is not practicable in animals, because of inherent limitations. The difficulty resides in the variable, but native, tendency of animals to compensate against the induction of experimental hyperparathyroidism, although never immunized to the extract. Despite this, and provided suitable animals and dosage are employed, it is possible with parathyroid extract to produce hypercalcemia, increased excretion of calcium and phosphorus through the urine, bone decalcification, the appearance of osteoclasts in Howship's lacunae in the course of the resorption, and fiber marrow. Whether the commercial parathyroid extract used in these experiments is actually the unchanged hormone of the parathyroid gland, one cannot say. Nor, again, can one determine at this time, the possible difference of this extract from the hormone elaborated by a hypersecreting parathyroid adenomatous tumor. The existence in man of a state of perverted mineral metabolism, as a prerequisite for the disease, appears to be excluded, as removal of the hypersecreting parathyroid adenoma alone apparently allows complete reversal of the pathologic state.

This brings us to the third division of our subject: the examination of some other diseases of bone in their relation to Recklinghausen's disease and the possible role of hyperparathyroidism in their evolution. The condition with which Recklinghausen's disease had most often been confused is genuine osteomalacia. There is no longer any real reason for these to be so mistaken. Anatomically, the bones in osteomalacia show very little marrow scarring, rela-

tively few osteoclasts, wide osteoid margins, and the absence of typical giant-cell tumors and cysts. Chemical analyses usually show—in contrast with Recklinghausen's disease—marked reduction in serum calcium and a negative balance for calcium, but the loss is chiefly through the stool. Furthermore, while the condition of the parathyroid glands may be completely normal in osteomalacia (which is never the case in Recklinghausen's disease), occasionally slight enlargement of all four parathyroids may be encountered, while even more infrequently one of these glands may reach a fairly large size. Erdheim indicated that even when the parathyroids are not grossly enlarged, microscopic evidences of hyperplasia may exist.

The distinction between Paget's disease and Recklinghausen's disease has been made very clear in recent years. The participation of the periosteum in the production of the pathologic lesions of Paget's disease is in contrast to the lack of periosteal activity in Recklinghausen's disease. Lamellation and thickening of the cortex of long tubular bones of the type seen in florid cases of Paget's disease do not occur in Recklinghausen's disease, in which the cortex is thinned. Giant-cell tumors of the nature of Recklinghausen's disease do not occur in Paget's disease. Microscopically, the bone transformed by Paget's disease shows the rather characteristic mosaic architecture. The parathyroid glands lack the tumorous enlargement that is seen in Recklinghausen's disease, and if there is any enlargement, it is but slight and generalized. The clinical examinations of the blood have shown no particular abnormalities in Paget's disease, except that the serum phosphatase tends to be very high, in fact, higher than in most other diseases of bone. Furthermore, studies of mineral balance have given no constant results in Paget's disease, while in Recklinghausen's disease there is a constant loss of calcium.

Generalized osteoporosis is an exceedingly common condition in adults. Vitamin deficiencies, endocrine disturbances, mineral dietary deficiencies, any one of a great number of causes may be responsible for the appearance of

marked demineralization. Very often, the underlying cause for the osteoporosis is so evident that no confusion whatever arises with Recklinghausen's disease. In senile osteoporosis, pronounced rarefaction of the skeleton may occur. Roentgenographic study of the bones, especially where the spine is deeply involved, may be misleading in the direction of Recklinghausen's disease. The pure microscopic lesion of senile osteoporosis is simply a chronic fatty atrophy of the bone. In an occasional and very advanced case of senile osteoporosis, small, single and confluent cysts—oily cysts due to liquefaction of fatty marrow—may be observed in some of the long tubular bones. In addition, more or less large brown areas containing hemosiderin but without giant-cells are occasionally seen and are referable to recent hemorrhage. The parathyroid glands, when studied, are often found to be normal; they have occasionally been reported showing enlargement recognized by the naked eye. Aside from the clinical suggestions, senile osteoporosis is not in the least related to Recklinghausen's disease and can be easily differentiated when it is considered in arriving at a diagnosis.

Furthermore, not all cases with generalized fibrocystic lesions of bone are necessarily Recklinghausen's disease. Unless such cases show the clinical and chemical evidences of Recklinghausen's disease, they are still to be included in the group for which the basis is as yet uncertain.

The parathyroids may or may not be enlarged in the conditions noted before. In other conditions, as clinical and experimental rickets, multiple myeloma, extensive carcinomatous metastases to the skeleton, the parathyroid glands likewise may or may not show enlargement. If parathyroid enlargement is present in any of the enumerated diseases, it does not appear, as in Recklinghausen's disease, in the form of a benign tumorous new growth. If encountered, one or as many as all four glands may be increased in size and weight. They are rarely, if ever, more than two or three times normal size. To establish hyperplasia of the parathyroids in any of these disturbances,

measurement of the size must be supplemented by proper histologic examination: this only can determine evidence of hyperplasia and unless it is adduced, hyperactivity of the glands, a priori, must be ruled out. It is often necessary to make serial sections of the parathyroid to demonstrate hyperplasia, and of even greater value is the staining of these sections for intracellular lipoid. Hyperplastic chief or principal cells show a lack or absence of intracellular lipoid.

Assuming that parathyroid hyperplasia is established in any such case, its place in the disease evolution is still indeterminate. Does this hyperplasia connote a hypersecretion, i.e., hyperparathyroidism? It may or it may not: if it does, the increased secretion of parathyroid hormone is secondary to the bone lesions or the disturbed state of mineral metabolism, and probably beneficial, rather than deleterious as it is in Recklinghausen's disease. Accepting the parathyroid enlargement in these conditions as associated with a hypersecretion, we have no way of proving such a state, as the only exact criteria for judging hyperparathyroidism are based on the pathologic effects of hypersecretion of the hormone. Functional or physiologic hyperparathyroidism can not as yet be measured. The very fact that the parathyroid hormone takes an important part in calcium metabolism in the normal person suggests that a disease causing a disturbance of mineral metabolism may secondarily provoke parathyroid hyperplasia and thereby hypersecretion. This may be interpreted as part of the attempt at adjustment or compensation to better control the mineral metabolism. Furthermore, subsidence of the original factors producing the parathyroid hyperplasia has, in certain proved instances, been associated with return of the parathyroids to normal size.

Now, the question of the rationale and basis of recent surgical popularity of parathyroidectomy in other diseases than Recklinghausen's is to be examined. This has been practiced especially in a group of conditions which

have been described under the title of "ankylosing polyarthritis". Reviewing some of the surgical literature that discusses this problem, one is struck by the multiform and often basically different conditions that are encompassed by this term. Included are instances of the Bechterew or Marie-Strumpell spine. This form of ankylosing spondyloarthritis may, of course, also be associated with stiffening of some of the larger joints—for instance, hips and shoulders. Then again, examples of the purely degenerative spondylosis deformans with osteophytic lipping and ankylosis of the vertebral bodies are also included under this vague title of "ankylosing polyarthritis", as are instances of hypertrophic arthritis and arthritis deformans tending toward or showing ankylosis.

The evidence that a so-called "parathyroidism" exists in these conditions, which can be corrected by removal of one or two parathyroid glands, rests on very tenuous testimony. They are: first, the raised serum calcium that is frequently reported found in "ankylosing polyarthritis" —the hypercalcemia reported as subsiding after parathyroidectomy; secondly, that these patients have enlarged parathyroid glands; and thirdly, that such patients are rapidly improved following parathyroidectomy. Critical examination of this data shows that the highest incidence of hypercalcemia has been reported from European clinics. The danger of obtaining misleading calcium values on technical grounds cannot be too greatly stressed. In this country, those who subscribe to the "parathyroidism" view for "ankylosing polyarthritis" have generally found normal calcium figures, but stressed the need of repeated examinations. Furthermore, if one accepts the results of Bauer as valid—and his work has surely been done under the most favorable auspices—then neither hypercalcemia nor disturbances in mineral balance occur in "ankylosing polyarthritis." Secondly, those who favor parathyroidectomy in "ankylosing polyarthritis", state that the removed parathyroids are enlarged and hyperplastic. Now, if one examines their published photographs, it is plainly evident

that the glands architecturally appear entirely normal—there is no evidence of adenomatous hypertrophy. Besides, they have failed to seek the more accurate indications of hyperplasia by staining for intracellular lipoid. Thirdly, the reason for the clinical improvement in such patients may be entirely unrelated to the parathyroidectomy. In a number of instances, where improvement has been reported following operative intervention, histologic examination has failed to disclose the presence of parathyroid tissue in the specimens removed. Besides, even when, as by Oppel, one complete thyroid lobe was removed, to insure partial parathyroidectomy, no improvement occurred in 16 of the 49 cases. It seems that any immediate improvement that does occur is possibly associated with rest in bed, and any permanent improvement to a remission or spontaneous subsidence through regression of the activity of the process. This generalization would seem to prevail against the value of parathyroidectomy in Paget's disease as well.

Parathyroid removal is, then, only clearly warranted in Recklinghausen's disease where a benign, tumorous adenoma exists.

METABOLIC DISTURBANCES IN RELATION TO THE TEETH*†

CHARLES F. BÖDECKER, D.D.S.

INTRODUCTION

The principal purpose of this paper is to call the attention of the medical profession to its responsibility in preventing dental disorders. Recent investigations indicate that dental health may sometimes be linked with general health, and that dental ailments may be symptoms of systemic derangements. Hence it is necessary that physicians in general and pediatricians in particular should evince more interest in dental problems.

General practitioners of medicine and dentistry who attempt to cover their vast fields are interested mainly in generalities; it is therefore the aim of the author to confine himself to these, and to leave the details for the study of those particularly interested in this subject (pediatricians and dentists). For these a fairly extensive bibliography is appended.

The teeth have been regarded until very recently as organs outside of the field of nutrition. As a result of such an erroneous conception, the field of dentistry has long been relegated to a group of specially trained men who combat the diseases of the teeth merely by reparative means. The only preventive treatment instituted to protect the dental mechanism against destruction has been an attempt to promote oral cleanliness. If such cleanliness is defined as a sterility of the oral cavity, then it can be regarded as futile.

The present form of dental service, inadequate as it is in really *preventing* dental destruction, is available to only a small part of the public. The proportion of the popula-

* From the laboratory of Histo-pathology, Columbia University School of Dental and Oral Surgery, New York.

† Delivered November 3, 1933.

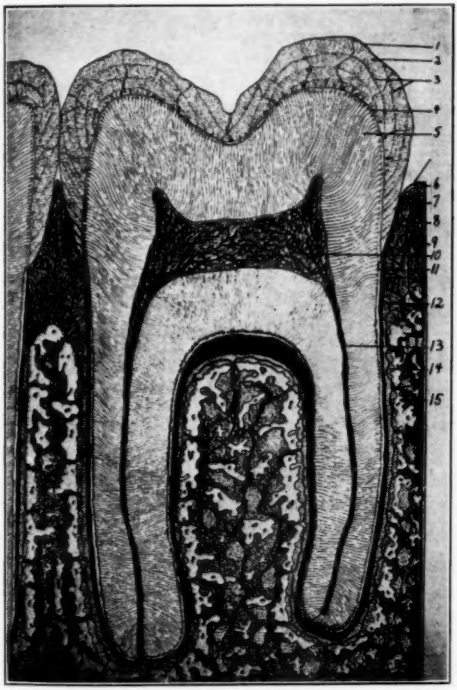
tion who receive dental care has been variously estimated to be no higher than twenty to thirty per cent. The reason why only symptomatic treatment has been resorted to by the dental profession as a whole is that systemic disturbances were thought to have no ill effect on fully formed teeth. It was believed and is still considered by many to be true that the calcified dental tissues receive no "nutrition" (added mineral salts), hence a disturbance of the nutritional balance of the body could have no ill effect on the teeth. Such erroneous views are very slowly giving way to a more sane, a more biological conception of the dental organs. Some health workers in general and the promoters of food products in particular, have filled the columns of periodicals and journals with commands to "feed your teeth." However praiseworthy this may be, a great deal of investigation must be undertaken before such an act can be understood scientifically, and furthermore, much work must be done to define the specific beneficial elements necessary to dental health. We know as yet too little of the histo-pathology of the dental tissues and almost nothing of their physiological activity. Dental physiology, as applied to the metabolism of the dental tissues, is in fact an almost non-existent science. With this in mind it is comprehensible that, as yet, little definite information can be given as to the relation of specific systemic disturbances to dental diseases.

Tissues of the Teeth

Some knowledge of the dental tissues is necessary for the comprehension of the manner in which metabolic disturbances may affect the teeth. A diagram, Figure 1, shows a longitudinal mesio-distal section of a lower molar and surrounding tissues. It is noted that the dentin forms the bulk of both the crown and the root of the tooth. Its formative and probably its nutritional organ, the dental pulp, or "nerve", occupies the interior of the crown and root; it is profusely supplied with blood vessels and nerves. The root dentin is covered with cementum which serves for the attachment of the root to the surrounding bone,

while the crown dentin is protected by enamel of varying thickness to withstand the force of mastication. It will be seen that each tooth can be regarded as a functioning unit, having its biological connection with the body through the vessels and nerves of the dental pulp. Incomprehensible as it may seem from a biological point of view, the dental pulp is believed by most investigators to have no function (except to form secondary dentin) after the completion of the tooth. In spite of the presence of a

Fig. 1. A Mesio-Distal Section of a Lower Molar, Alveolus and Gingival Papillae.*



1. Position of enamel cuticle.
2. Enamel rods showing gnarled character.
3. Striae of Retzius, incremental lines on enamel.
4. Dentino-enamel junction.
5. Tubules in the dentin.
6. Epidermis.
7. Old idea of gingival crevice.
8. Dermis of the gingiva.
9. Blood supply.
10. Dental pulp with profuse blood supply.
11. Granular layer of Tomes, situated only in the root, covered by cementum.
12. Cementum, thinnest at the cervix; thickest at the bifurcation and apices of the roots (solid black).
13. Pulp canal.
14. Peridental membrane attaching the root of the tooth (cementum) to the surrounding alveolar bone.
15. Lamina dura of the alveolus frequently perforated for the passage of blood vessels to supply the peridental membrane.

* From Charles F. Bödecker, "Elementary Histology for Dental Hygienists." New York, 1933. John Felsberg, Inc.

profuse blood and nerve supply in the dental pulp, it is believed that the calcified dental tissues, particularly the enamel, have no connection with the body. This erroneous view seems to be held by all except Beust[1], Fish[10], and the author[2,3]. The newer researches, however, are forcing more and more investigators to accept a saner biological conception.

An examination of Figure 1 shows that the enamel, covering the entire exposed portion of the tooth, is a protective tissue to prevent destruction from the outside. The resistance of the tooth, particularly of a young person, is therefore primarily dependent upon the ability of the enamel to withstand attack of the acid which is supposed to be active in dental caries. Evidence is accumulating to show that the resistance of the enamel (and also that of the dentin) against external attack is dependent upon systemic conditions. When this is finally proven, the varying activity of dental caries will become more clear. The entire subject is now under active investigation by the Columbia University Dental Caries Research Group, financially assisted since 1930 by the Commonwealth Fund of New York.*

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* The Commonwealth Fund Grant for the Study of the Cause of Dental Caries:

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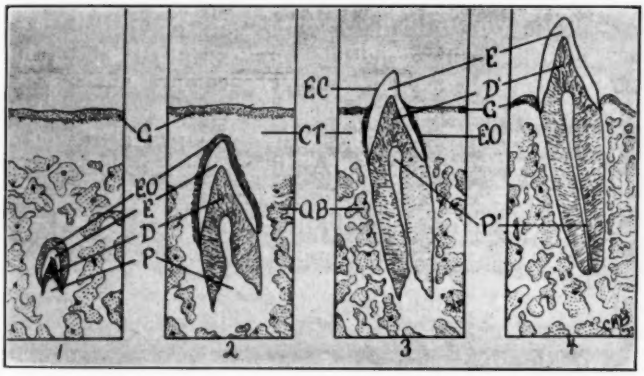
Mention must be made of the manner in which the teeth are developed, for the proper understanding of the metabolism of the dental tissues. A comprehension of the development of the enamel is particularly essential in this respect. This structure is formed by the enamel organ of ectodermal origin. Enamel growth of the teeth begins at the dentino-enamel junction and progresses outwardly, so that the last formed enamel is on the surface of the tooth (Figure 2, diagrams 1 and 2E.) The tooth then

Fig. 2. Stages in the Eruption of a Tooth.



The enamel organ is destroyed when the tooth pierces the gingiva, hence it is believed that the enamel requires no further mineral salts.

G—Gingiva.	D—Dentin.	EC—Enamel Cuticle.
EO—Enamel Organ.	P—Pulp.	CT—Connective Tissue.
E—Enamel.	P_1—Dental Papilla.	AB—Alveolar Bone.

pierces the gum tissue in order to become a functioning unit of the oral cavity and thus the enamel organ is destroyed. (Figure 2, diagrams 3 and 4.) Its remnant forms the enamel cuticle (EC) which, being cut off from all blood supply, can no longer function; thus no new enamel can be developed after the eruption of the tooth. *It is for this reason* that the mistaken conviction has so

long existed that systemic disturbances cannot affect the enamel by means of the circulatory system and that therefore fully formed teeth can be acted upon only from the exterior; *i.e.,* by the saliva, bacteria, and by food retention in the oral cavity. This belief has long been a barrier to the investigation of the possible systemic causes of dental caries.

A further strengthening of this erroneous belief was the conviction that this tissue is a purely mineral structure, containing no protein matrix. If this were true, then it would be impossible for lymph, originating from the dental pulp, to penetrate the enamel. As has been stated previously, the young functioning tooth was considered to be completely matured and no mineral salts ("nutrition") were needed after this time. The comprehension of the attitude of most investigators of teeth is necessary for a thorough understanding of the problem of dental caries. Hence it is essential to recapitulate the incorrect conception of the enamel held by many investigators even today; this is that the enamel of fully formed teeth can not be affected from within by general metabolic disturbances because:

a. Its formative organ is destroyed on the eruption of the tooth.

b. It is an inorganic structure, containing no protein matter which might serve as channels for the distribution of mineral salts.

c. It is therefore impermeable to a possible calcifying or decalcifying lymph which might originate from the blood stream by way of the dental pulp and the dentin.

For these reasons, the enamel was considered to be completely matured when the tooth pierced the gum, and therefore systemic disturbances, whatever their nature, could not affect the "nutrition" of the enamel. In line with this reasoning, it was thought that a deleterious influence could affect the enamel only from the exterior.

This view is slowly losing ground as:

a. The enamel has been shown to contain a protein matrix, which may include channels for the transportation of lymph; this matrix is more abundant in youth than in later adult life. [Bödecker[3], Gies[4], Malleson[5], Bibby[6].]

b. The enamel, under certain conditions, has been shown to be permeable to dyes, etc., in vitro and in vivo. [Bunting and Rickert[7], Beust[8], Klein and Amberson[9], Fish[10], Bödecker[11,12], Applebaum[13,14].]

c. The enamel has been shown to undergo a progressive hardening after the eruption of the teeth. [Karlström[15].]

d. The enamel shows structural variations in calcification as defined by the Grenz-ray (soft x-ray). [Hollander, Applebaum and Bödecker[16].]

All these observations support the view that the enamel is permeated, at least in children and young adults, by a lymph which may increase its hardness, and protect it against the external destructive products of bacteria and decomposing food debris. It is possible therefore that dental caries, which is most prevalent in children and young adults, may be due to a failure on the part of the body to mature the teeth thoroughly after their eruption and to build up sufficiently their resistance to external attack.

The irritating cause of dental caries is probably an acid, formed by the decomposition of food debris around the teeth; this we term the *environmental* (exciting) cause of caries. This factor, however, does not complete the picture of the disease; a predisposing factor seems also to be present.

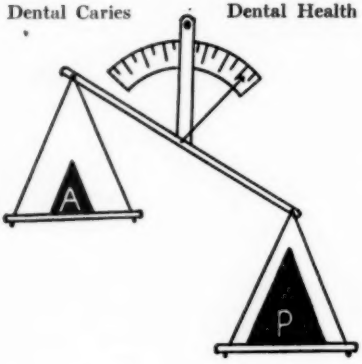
Balanced against the environmental causes of dental caries, there appears to be a *protective* mechanism in each tooth (the dental pulp), which increases the resistance of the dental tissues to attack. Figure 3 a, b, c, expresses the

Fig. 3. Diagrams to illustrate the two opposing factors active in dental caries. The exciting cause of the disease is the fermentation of food debris and other factors affecting the teeth from the *exterior*. This is shown in the diagrams as the attacking factor (A). Opposed to this is the protective factor (P), a defense mechanism active from the *interior* of the tooth; its activity is dependent on systemic conditions.

Fig. 3a.

HIGH IMMUNITY TO DENTAL CARIES

(Primitive Races)



Dental Attacking Factor

(A)

Weak: **due to natural diet and intense use of teeth; oral prophylaxis unnecessary.**

Dental Protective Factor

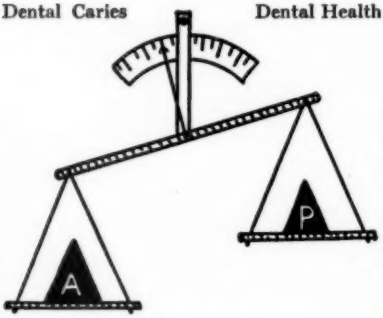
(P)

Strong: **due to good general health.**

Fig. 3b.

SLIGHT SUSCEPTIBILITY TO DENTAL CARIES

(Average Civilized Persons)



Dental Attacking Factor

(A)

Strong: **due to adhesive civilized diet resulting in considerable food retention; oral prophylaxis brings dental caries to a standstill.**

Dental Protective Factor

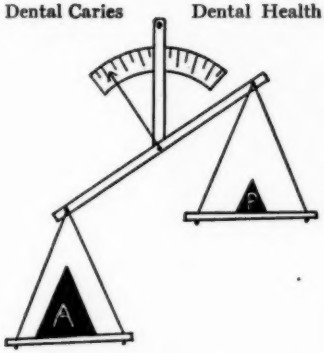
(P)

Weak: **due to a reduction of vitality of the body (unbalanced diet, lack of sunshine and exercise).**

Fig. 3c.

GREAT SUSCEPTIBILITY TO DENTAL CARIES

(Persons suffering from systemic disturbances)



DENTAL ATTACKING FACTOR

(A)

Very strong: due to excessive food retention resulting from disuse of painful teeth; oral prophylaxis useless, due to weak dental protective factor.

DENTAL PROTECTIVE FACTOR

(P)

Very weak: due to metabolic disturbances.

view of the author concerning the mutual relationship of these two major factors active in dental caries. The proper functioning of the protective mechanism is dependent largely on optimal systemic conditions. It is therefore clear that the attacking factor (food debris, bacteria, etc.) is counterbalanced by the protective factor. Thus a reduction of the natural protection of the teeth, through systemic disturbances, results in an increased activity of dental caries, unless it is counterbalanced by a more strict oral hygiene; frequent use of the tooth brush removes the food debris around the teeth and thus reduces the formation of oral acid (Figure 3b).

Civilization has changed our diet as well as our habits, so that our physical resistance has been lowered, in comparison to that of primitive people. Thus the teeth have lost their natural protection and we must resort to artificial means of cleansing to reduce the environmental causes of caries (food debris, bacteria, etc.), in order to combat dental destruction. This brief description of the two opposing factors of dental caries explains the necessity for scrupulous oral hygiene in *civilized* people, for most of us have a lowered resistance to dental caries.

Persons with rampant caries (Figure 3c) have such an exceedingly low resistance (P) that no amount of brushing of the teeth can prevent their decay.

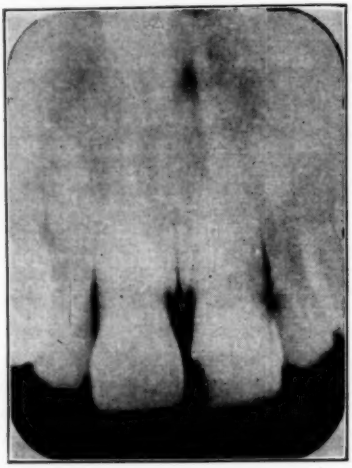


Fig. 4. X-ray of two central incisor teeth which shows that saliva, food retention, and bacteria cannot be the only factors in the cause of dental caries, as only one of the two closely adjoining tooth surfaces has become carious.

The findings of the Agnews[17], Boyd and Drain[18], Eddy[19], Percy Howe[20,21], Hanke[22], Martha Jones[23], Marshall[24,25], McBeath[26], Klein and McCollum[27,28], May Mellanby[29,30,31], Price[32,33,34], and others show that dental disorders may be greatly reduced by a proper adjustment of the diet. Some of these investigators maintain that a lack of vitamin C is principally responsible for the activity of dental caries (Howe, Hanke). Weston Price regards vitamin B and mineral salts as the important elements in a caries free diet. Some believe that the lack of vitamin D is the offending factor (Mellanby). Finally, a disturbance in the cal cium-phosphorus balance (which includes vitamin D) is the factor to which most recent investigators point as being

responsible for the high activity of dental caries. This conception is held by the Agnews, Jones, Marshall, Klein and McCollum, as well as by the Columbia University Dental Caries Research Group. The report of this group to the Commonwealth Fund, March, 1933, contains among much other material this statement:

"It is clear from these experiments that systemic factors associated in some way with the calcium-phosphorus metabolism are definitely related to the resistance of the teeth to decay. Whether other systemic factors exert an influence remains for future investigations to determine."

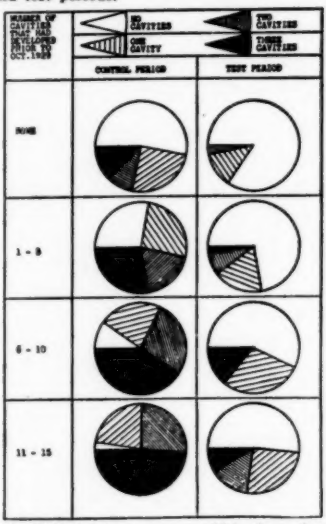
The sources of vitamin D appear to be an important factor in the prevention of dental caries, as shown by the observations of McBeath, of our group. This investigator was able to reduce dental caries to one-ninth its activity by irradiating children, ranging in age from 8 to 14 years, with ultra-violet light; that is, *dental caries was nine times more active in the control group than in the experimental group.* Mellanby and Jones believe that a diet, composed largely of cereals, is harmful, inasmuch as they have observed a marked increase of dental caries under these conditions. Hanke (Chicago Dental Research Club) has recently published an extensive report concerning "Nutritional Studies on Children[22]." Their results are typical of the work of other investigators in reducing dental caries by dietary means. Observations were made on 323 children over a period of two years; the results are shown in a copy of one of their graphs (Figure 5).

Discussion

The foregoing brief report of experiments to control dental caries by dietary means shows that it is possible, in the majority of children, to reduce and even in certain cases to prevent, this very common destructive dental disease. The divergence of opinion, concerning the specific beneficial factors, active in maintaining dental health indicate that our problem is not yet solved. Furthermore, the harmful elements of the diet which seem to exist, have

Fig. 5.

Correlation of the number of cavities that had developed to October, 1929 (total previous decay) with the incidence of dental caries during the control and test periods.



Each circle is divided into segments which show the percentage of children in which no, one, two and three (or more) cavities developed during the period designated above. The number of cavities that had developed prior to October, 1929, appears at the left. Read circles from left to right to determine changes occasioned by a change in diet. Read circles from top to bottom to determine variations in the incidence of dental caries associated with an increasing tendency toward this disease.

From "Nutritional Studies on Children," M. T. Hanke, et al, The Chicago Dental Research Club, *Dental Cosmos* **75**, 742, August, 1933.

not yet been definitely specified. An excess of carbohydrates is still held by some investigators to be partly responsible for an increased activity of dental caries. The work of Mellanby and Jones indicts cereals as the principal cause of the great activity of dental caries. Cereals have been fed commonly to generations of children, and it is possible that this type of diet, which forms an acid ash in the body, is harmful to the teeth and explains at least in part the intense activity of dental caries in the young [Mellanby[30], Jones[23] and Kugelmass, King and Bödecker[35]]. Kugelmass expresses a definite opinion on this subject: "children maintained on ketogenic diets, *base-forming* in

their mineral content, showed low life caries indices, while those maintained on ketogenic diets, *acid-forming* in their mineral content showed marked dental caries." Further that: "children free from dental caries showed consistently a dietary intake excessive in alkali-forming minerals, with a preponderance of raw fruits and vegetables."

The following quotation of Hanke[22] is interesting inasmuch as it indirectly throws light on the relation of the teeth to the body. Hanke says: "It is becoming increasingly evident that dental caries can be influenced in some cases by modifying the diet. The change in diet must, then, either lead to a change in the oral condition or lead to a change in the tooth itself. It is impossible at present to determine just why dental caries should be arrested by a change in diet. An incontrovertible proof as to whether the change occurs in the oral cavity or in the tooth itself is not contained in our studies nor in those of other investigators to which we have had access."

The results of McBeath[26] are particularly instructive in this respect, inasmuch as the beneficial effect of ultra-violet light rays does not reach the teeth by way of the mouth, but must affect primarily the systemic condition, which in turn improves the resistance of the dental tissues to caries. All forms of dietary additions such as orange juice, cod liver oil, vegetables, fruits, etc. come into direct contact with the teeth and therefore might be thought to increase their resistance to dental caries by means of this contact. But this condition does not apply to the observations of McBeath concerning the effect of ultra-violet light. It is thought by many investigators that the saliva is the beneficial or harmful agent affecting the teeth during varying systemic conditions, but Figure 4 shows that this can not always be true. In this x-ray we note that caries has attacked only one of two closely adjoining teeth. Saliva can not be made responsible for attacking one tooth and protecting the other.

The fact therefore that teeth may be benefitted by ultra-violet irradiation is an indication of their biological con-

nection with and dependence upon the body. The frequent reiteration of this statement may seem needless to an audience composed principally of biologically-minded medical practitioners, nevertheless it is necessary, in order to break down the prejudice under which most investigations of dental caries have been carried on.

A study of the recent literature on the subject of dental caries shows marked contradictions. Some investigators point to the advantages of a diet composed chiefly of fruits and vegetables and stress the benefits of ultra-violet light. They strengthen their argument with examples of the excellent teeth noted in some primitive races living in the tropics. Contradictory evidence in this respect is presented by the Eskimos, who live six months in darkness and subsist on an entirely different diet, a high protein diet. Their teeth are in an excellent condition so long as the race is not exposed to our civilized diet [Waugh[36]]. These two contradictory observations are so confusing that some people have disregarded diet as a major factor in the cause of dental caries. However, Martha Jones suggests a rather ingenious explanation of this paradox. She believes that an abundance of ultra-violet light, such as people of the tropics enjoy, results in a slight acidosis, hence such people require a highly alkaline diet, namely fruits and vegetables to maintain the normal balance. The Eskimos, on the other hand, living in a region where there is a scarcity of ultra-violet light, tend as a result to an alkalosis; hence they require a higher acid ash diet in order to counteract this alkalosis. This view appears plausible, for it is a common practice of dietitians to advise the reduction of meat consumption during the summer months in the temperate zones, and the increase of the protein content of our diet during the winter months. Dr. Jones is continuing her researches in this direction and if her views are correct, it will clear our conception of the type of food necessary to maintain dental health under different climatic conditions; and thus the apparent paradox of dentally perfect races living on radically different diets will be removed.

Dental caries is undoubtedly due to a number of causes. The author suggests that the relation of the teeth to systemic disturbances may be expressed tentatively in the diagram shown in Figure 6. This chart is presented merely to crystallize our thoughts on the problem. If this theory, showing the complexity of the causes of dental caries is approximately correct, we begin to understand why the views of different investigators are so widely divergent, and to comprehend that each may be correct in his individual observations made in localized districts. For instance, the activity of dental caries in one locality may be due to a lack of sufficient mineral salts resulting from an impoverished soil, as is claimed to be the case in South Africa. [Lennox[37]] In a second locality, dental caries may be caused by a lack of sufficient sunshine; in a third, to an absence of one of the vitamins. Naturally each observer believes that the deficiency he observes is the sole cause of dental caries; and he may be correct so far as his locality is concerned. The chart (Figure 6) shows a number of deficiencies, each of which may be regarded as a contributing factor in dental caries, since such a deficiency can disturb the metabolism of the body. This in turn reacts on the dental pulp, the nutritional organ of the tooth. A disturbance of the function of this organ results in a derangement of the protective mechanism of the tooth; this constitutes the predisposing cause of dental caries. It is only when the natural protection of the teeth is lowered through an improper functioning of the dental pulp that the exciting causes of dental caries can become active (food debris, bacteria).

All investigators who have observed nutritional experiments have noted that the activity of dental caries is reduced under improved nutrition. But the fact that this disease can not be prevented in all cases, again shows that our understanding of the problem is by no means complete. We do not yet comprehend the mechanism of destruction of the dental tissues during the feeding of an unbalanced

diet; neither can the manner be agreed upon in which the teeth are protected during the period of proper nutrition. This protective dental mechanism whose disfunction allows the teeth to be ravaged by dental caries, must first be defined by the histologist and physiologist in order to make further progress in the solution of the problem of dental caries. When that is achieved, attempts will be made by the chemists to analyze the dental lymph and to find differences in the constitution of this fluid in caries-immune and in caries-susceptible persons. Then, knowing the elements necessary to maintain a high resistance of the teeth to decay, we may possibly administer these elements to the body, at the same time stimulating the organs responsible for their assimilation. It will be important also to define those food elements affecting body fluids which result in systemic disturbances permitting the decalcification of the teeth; when these are recognized, the medical profession can warn the public of the dangers of an improper diet, and can stimulate public interest in individual responsibility in maintaining general health for the possession of sound teeth.

Conclusion

Recent findings indicate that a disturbance of the calcium-phosphorus balance exerts a deleterious influence on the teeth, making them more prone to dental caries. In the light of this knowledge, it seems necessary that dental caries should no longer be treated solely by reparative means but that true prevention, by systemic treatment, should be instituted.

The teeth are often so seriously affected by caries during childhood that a number must be removed; thus such persons go through life with an impaired dentition, or, if pulp removal is resorted to, one or more possible sources of dental foci of infection are present. The dangers of focal infection may have been exaggerated by some, nevertheless there is still a real danger and this danger should be faced squarely.

The only certain way that dental foci of infection can be eliminated in the future and at the same time insure all persons a full complement of teeth, is to *prevent* the inception of dental caries.

In the future, the principal duty of the dentist will be, as it has been in the past, the repair of the ravages of dental caries and pyorrhea alveolaris. On the other hand, the responsibility for the *prevention* of dental disorders will rest on the shoulders of the medical profession. In this connection the dentist will be able to perform another valuable service, namely that of noting oral disturbances which may be reflected from incipient systemic disorders. Thus, the dentist who sees his patients in a state of comparative good health, can warn them of impending trouble and suggest a consultation with the physician. Observant dentists have frequently noted that their reparative dental work does not keep pace with the rate of dental destruction, and in despair they sometimes suggest to the patient a consultation with the family physician. This action is definitely the duty of the dentist. However, an attempt to prescribe should not be made by the dentist. This should be done only by the physician after a thorough physical examination in order to define the deficiency existing in the case of each patient.

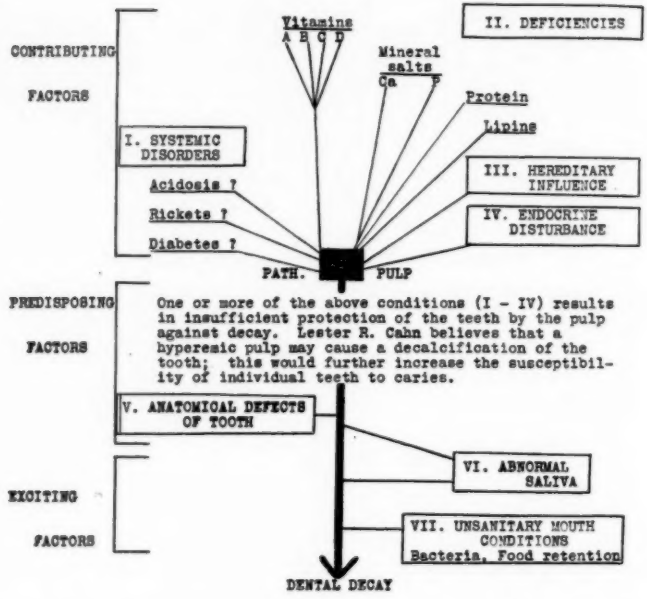
For a better understanding of dental disorders having a systemic origin, it may be advisable in future to create a new specialty covering particularly the borderline of dentistry and medicine. Even though it is uneconomic to train all dentists as medical graduates, it is advisable, nevertheless, to instruct a selected group of men in both fields of medicine and dentistry. Such men will be able to act as consultants and to suggest systemic treatment of general disturbances which are suspected of causing oral diseases.

There is evidence that rampant dental caries is a symptom of some systemic disturbance and it is suspected that pyorrhea alveolaris can be regarded in the same light. I have frequently referred a person to a physician for a

Fig. 6.

SOME POSSIBLE FACTORS INVOLVED IN THE CAUSE OF DENTAL DECAY

MOST ACTIVE DURING YOUTH



physical examination, and in many cases the condition of the patient was pronounced normal. However, in some instances, systemic disturbances were diagnosed some time after the first consultation. This leads me to believe that our laboratory tests, particularly in relation to the blood and the endocrines, are not sufficiently delicate to define some as yet unknown variation from the normal, which may exist in incipient systemic disturbances. The teeth, being farthest removed from the source of general nutrition, probably suffer first as a result of certain metabolic disturbances. Hence it is my belief that the teeth may at some time in the future be regarded as a delicate indicator of some phases of bodily health, even before a physical examination discloses any systemic derangement. This

view is by no means new, it was generally maintained during the last decades of the nineteenth century, but has been since disregarded as a result of the belief that the welfare of the teeth was not dependent upon bodily conditions.

Consequently, if we wish to prevent dental caries in children and young adults, and pyorrhea alveolaris in the middle aged and the old, systemic treatment must be instituted. For the purpose of defining such a mode of treatment, a more intimate association of physicians and dentists is necessary.

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DEATHS OF FELLOWS OF THE ACADEMY

BROWN, JAMES SPENCER, M.D., Pinehurst, North Carolina; graduated in medicine from the College of Physicians and Surgeons, New York City, in 1885; elected a Fellow of the Academy June 6, 1895; died, August 18, 1934.

FISHBERG, MAURICE, M.D., 1136 Fifth Avenue, New York City; graduated in medicine from New York University in 1897; elected a Fellow of the Academy January 2, 1913; died August 30, 1934. Dr. Fishberg was a Fellow of the American Medical Association and a member of the County and State Medical Societies, the New York Academy of Sciences, the American Ethnological Society, the American Anthropological Association and the American Folklore Society. Dr. Fishberg was Chief of the tuberculosis service at Montefiore Hospital. Besides treatises and articles on pulmonary tuberculosis, Dr. Fishberg wrote extensively on anthropological subjects and had made a special study of the Jewish populations of various countries.

KILBANE, EDWARD FRANCIS, M.D., 40 East 61 Street, New York City; graduated in medicine from Cornell University Medical College in 1901; elected a Fellow of the Academy March 7, 1912; died, August 18, 1934. Dr. Kilbane was a Fellow of the American Medical Association and the American College of Surgeons, a member of the County and State Medical Societies, the American Urological Association and the Alumni Association of Lying-in Hospital. He was Associate Surgeon at Roosevelt, Genito-Urinary Surgeon at Misericordia, Consulting Urologist to Tuxedo and Flushing, and Urologist to Lincoln and St. Mary's Child Hospitals.

TIECK, GUSTAV JOHN ERIC, M.D., 2 East 54 Street, New York City; graduated in medicine from the Long Island College Hospital in 1899; elected a Fellow of the Academy October 6, 1910; died, August 18, 1934. Dr. Tieck was a Fellow of the American Medical Association, the American College of Surgeons and a member of the County and State Medical Societies. He was Director of otolaryngology at Broad Street Hospital.

WAINWRIGHT, JONATHAN MAYHEW, B.A., M.D., M.A., 327 North Washington Avenue, Scranton, Pennsylvania; graduated in medicine from the College of Physicians and Surgeons, New York City, in 1899; elected a Fellow of the Academy April 7, 1904; died, August 3, 1934. Dr. Wainwright was a Fellow of the American Medical Association, the American Surgical Association, the American College of Surgeons, and a member of his County and State Medical Societies. For many years Dr. Wainwright had been Surgeon in Chief of the Moses Taylor Hospital in Scranton. During the World War he commanded a base hospital in France. In his later years he was especially interested in cancer research and since 1930 had been President of the American Society for the Control of Cancer.

OFFICERS OF SECTIONS AND AFFILIATED SOCIETIES, 1934-35

Dermatology and Syphilology, 1st Tuesday

Chairman	*Secretary*
Ray H. Rulison	E. William Abramowitz
145 East 54 Street	853 Seventh Avenue

Surgery, 1st Friday

Guilford S. Dudley	Condict W. Cutler, Jr.
27 East 63 Street	667 Madison Avenue

Neurology and Psychiatry, 2nd Tuesday

C. Burns Craig	Leon H. Cornwall
Neurological Institute	30 East 76 Street

Historical and Cultural Medicine

2nd Wednesday of November, January, March and May

Russell L. Cecil	Jerome P. Webster
33 East 61 Street	180 Ft. Wash. Ave.

Pediatrics, 2nd Thursday

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132 East 71 Street	107 East 85 Street

Ophthalmology, 3rd Monday

Webb W. Weeks	W. Guernsey Frey, Jr.
20 East 53 Street	121 East 60 Street

Medicine, 3rd Tuesday

Randolph West	Paul Reznikoff
622 West 168 Street	N. Y. Hospital, 525 East 68 Street

Genito-Urinary Surgery, 3rd Wednesday

Augustus Harris	Joseph A. Hyams
306 Park Place, Brooklyn	78 East 79 Street

Otolaryngology, 3rd Wednesday

Marvin F. Jones	Wm. Wallace Morrison
121 East 60 Street	39 East 50 Street

Orthopedic Surgery, 3rd Friday

Leo Mayer	Walker E. Swift
140 West 79 Street	115 East 61 Street

Obstetrics and Gynecology, 4th Tuesday

Harvey B. Matthews	Walter B. Mount
643 St. Marks Ave., Brooklyn	11 Seymour Street, Montclair, N. J.

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Society for Experimental Biology and Medicine, 3rd Wednesday

President	*Secretary*
A. R. Dochez	A. J. Goldforb
136 East 67 Street	City College, Convent Ave. & 139 St.

Harvey Society, 3rd Thursday

R. Keith Cannan	Edgar Stillman
Bellevue Hospital	22 East 69 Street

New York Pathological Society, 4th Thursday

Paul Klemperer	Irving Graef
385 Central Park West	Bellevue Hospital

New York Roentgen Society, 3rd Monday

A. L. Loomis Bell	C. Wadsworth Schwartz
L. I. Hospital, Brooklyn	33 East 68 Street